A SUMMARY OF THE SYMPTOMS IN SIXTY-ONE CASES OF LOCOMOTOR ATAXIA, WITH ADDITIONAL REMARKS,[1]

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The sixty-one cases of locomotor ataxia, the most important symptoms of which are here given in the order of their relative frequency, have been examined and treated by the writer in sanitariums with which he has been connected. These patients were all males, which is quite uncommon for so large a number of cases. The percentage of females suffering with this disease is, however, usually small.

A history of syphilis was given in thirty-one cases out of forty-nine. Of the remaining eighteen cases of the forty-nine, fourteen had had gonorrhœa, been excessive in sexual indulgence, or gave other evidence of possible exposure to syphilis. Of the remaining twelve cases of the sixty-one, syphilis was either denied, or this point was not determined in the history of the case. In the cases that gave a history of syphilis, from two years to thirty years intervened between the primary venereal disease and the initial symptoms of locomotor ataxia. In most cases the initial symptoms of ataxia appeared from eight years to fifteen years after syphilis had developed. In two cases the disease followed soon after a mechanical injury. A history of exposure to wet and cold was given in seven cases. One case developed immediately after an attack of typhoid fever. This last case, however, differed from cases usually seen, in that the disease was not progressive in character, and the only prominent symptoms present were well marked ataxia in all four extremities, as well as in the muscles of speech.

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[1] Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

The majority of these cases came from the middle and higher walks of life. In twenty-nine cases, the disease first showed itself between the ages of thirty and forty years; in twenty cases, between the ages of forty and fifty years; in six cases, between the ages of fifty and sixty years. In two cases the disease began at the age of twenty-five, and in one case, following typhoid fever, it began at the age of twenty-two years. The initial symptom, as given by patients in the history of their cases, was as follows:

In thirty-seven cases the initial symptom was pain in some part of the body, and was usually described as "rheumatic"; in three cases, it was gastric crises; in three cases, laryngeal crises; in four cases, incoördination of the lower limbs. In the remaining cases the initial symptom consisted of one or more of the following: Various paræsthesias in the extremities, diplopia, partial blindness, vertigo, difficulty in emptying the bladder, loss of sexual function, general feeling of weakness—frequently without exertion—and digestive disturbances.

The symptoms of the sixty-one cases tabulated in the order of their frequency are as follows:

1. Knee-jerk absent in......................55 cases.
2. Severe paroxysms of pain in..............54 cases.

In twenty-one of these cases severe pains were in the arm, trunk and legs; in fifteen, in the legs alone; in thirteen, in the trunk and legs; in one, in the arms alone; and in one, in the trunk alone.

3. Ataxia in locomotion, with eyes closed (56 cases examined), present in.............54 cases.
4. Various paræsthesias, as numbness, pricking, formication, etc., present in.............53 cases.

In thirty of these cases, paræsthesia was confined to the lower extremities; in twenty-one, to both lower and upper extremities; in two, there was paræsthesia in the face, as well as in the lower and upper extremities.

5.	Static ataxia, with eyes closed (56 cases examined), present in	54 cases.
6.	Anæsthesia (referring to tactile, temperature and pain sense, one or more being affected), present in	45 cases.

In fourteen of these, the anæsthesia was in the upper and lower extremities; in two, in the trunk and upper and lower extremities; in two, in the face and upper and lower extremities; in the remainder, in the lower extremities.

7.	Girdle sensation about trunk present in	39 cases.
8.	Constipation present in	39 cases.
9.	Static ataxia, with eyes open (56 cases examined), present in	41 cases.
10.	Ataxia in locomotion, eyes open (56 cases examined), present in	41 cases.
11.	Cold extremities in	34 cases.
	Loss of sexual power, partial or complete, in	35 cases.
	General feeling of weariness, with diminished motor power, in	37 cases.
12.	Area of hyperæsthesia about abdomen and lower part of trunk, with increased skin reflexes in the same area, in	29 cases.
13.	Indigestion and stomach disorders in	33 cases.
	Considerable loss of weight in	33 cases.
14.	Myosis present in	28 cases.
	Argyll-Robertson pupil (that is, pupil contracted, responding to light, but not to accommodation), present in	28 cases.
15.	Nervous irritability and despondency in	25 cases.
16.	Accelerated pulse (85 or above) in	25 cases.
17.	Skin reflexes (plantar, cremasteric and abdominal) increased in	24 cases.
	Incomplete retention of urine in	27 cases.
18.	Insomnia in	20 cases.
19.	Ataxia in arms in	15 cases.
20.	Diplopia usually present at beginning of disease in	14 cases.
	Skin reflexes absent in	16 cases.
21.	Delayed sensations in	14 cases.
	Partial incontinence of urine in	15 cases.
22.	Ptosis (transient or permanent) in	13 cases.
	Attacks of sudden vertigo in	13 cases.
	Partial iridoplegia in	11 cases.

23.	Partial deafness in	13 cases.
24.	Plantar skin reflexes absent, or very much diminished, with other skin reflexes normally present, in	10 cases.
	Gastric crises in	11 cases.
25.	Tenderness along the spine in	7 cases.
	Diminished faradic irritability of muscles in	6 cases.
	Diminished galvanic irritability in	6 cases.
	Unable to walk with crutches or two canes in	6 cases.
26.	Rise of temperature during paroxysms of pain in	6 cases.
	Optic nerve atrophy in	7 cases.
	Laryngeal crises in	6 cases.
	Diarrhœa in	8 cases.
	Pupils dilated in	8 cases.
27.	Unequal pupils in	6 cases.
28.	Polyæsthesia in	4 cases.
29.	Complete retention of urine in	3 cases.
	Complicated with marked muscular atrophy with the electrical reaction of degeneration in muscles in	2 cases.
	Tremor in hands in	2 cases.
	Anæsthesia in face in	2 cases.
	Taste affected in	2 cases.
	Area of hyperidrosis about trunk in	2 cases.
	Knee-jerk present and normal in	2 cases.
	Knee-jerk present, but unequal, right stronger, in	1 case.
30.	Knee-jerk exaggerated in	1 case.
31.	Hyperidrosis of both legs in	1 case.
	Knee-jerk present, but diminished, in	1 case.
	Knee-jerk present in one limb and absent in the other	1 case.
	Complicated with general paresis in	1 case.
	Traces of albumin in urine in	1 case.
	Complete incontinence of urine in	1 case.
	Traces of sugar in urine in	1 case.

With reference to the symptomatology of tabes, I believe that this is fairly well represented in a report of the symptoms found in sixty-one given herewith. Many of these are seldom noticed, or only lightly touched upon, by the majority of writers on this subject; and they are even more frequently overlooked in examinations of patients suffering with this disease.

The symptoms apt to be overlooked in connection with this disease are an exaggeration of the skin reflexes, partial deafness, and accelerated pulse. In addition to these, there is in many cases insomnia, considerable loss of weight, with more or less disturbance of the digestive organs, and very frequently a relaxed condition of the abdominal muscles, allowing the stomach and bowels, and sometimes other abdominal organs, to become prolapsed and displaced. This is often a very marked condition in locomotor ataxia.

Another fact that may be noticed in this connection is that the knee-jerk is not always absent in locomotor ataxia, as is usually supposed. Of these sixty-one cases reported, it was found absent in fifty-five. Of the six remaining cases, it was present, and apparently normal, in two; present but unequal, the right being the stronger, in one; present, but much diminished, in one; present in one limb and absent in the other, in one case; and in one case it was certainly exaggerated. There is another class of cases of so-called ataxic paraplegia, in which the knee-jerk is exaggerated; but in this class there is an actual paralysis present in the lower limbs, in addition to the symptoms of locomotor ataxia, and the lesion in the cord occupies a larger and entirely different territory than in typical locomotor ataxia. The case here referred to, in which the knee-jerk was exaggerated, does not belong to this class. In this particular instance there was no paralysis of the lower limbs, and a careful study of the symptoms of the case would in no way warrant putting it in this class of so-called ataxic paraplegias.

Dr. S. Weir Mitchell, of Philadelphia, has reported a case in which the knee-jerk was exaggerated, but the symptoms of ataxia were in the upper extremities entirely.

Where the anæsthesia affects the arms and hands, I have found in several cases that it was confined to the ulnar nerve. This observation has been made by others.

With reference to the exaggeration of the skin reflexes, in my own examinations I have found this condition in many cases which I have had an opportunity of examining at an early stage of the disease. As the malady progresses, the skin reflexes diminish, and finally disappear entirely, so that in most cases of tabes in the second or third stage of the disease the skin reflexes are entirely absent. I have found this exaggeration of the skin reflexes of great diagnostic value. Cases are frequently seen where, in the early stages of the disease, it is somewhat difficult to make an accurate diagnosis. The symptoms are not prominent, and, perhaps, a diagnosis must be formed upon only a general history of the case, together with two or three symptoms. In instances of this kind, when I have found the skin reflexes exaggerated, in connection with a few other symptoms pointing to this disease, I have usually had no trouble in making a diagnosis of locomotor ataxia.

I remember distinctly one case coming under my observation, where the only symptom pointing to incipient locomotor ataxia, with only the very slightest suggestion of incoördination, was decided skin reflexes, particularly plantar. The knee-jerk was present, and all other symptoms of the disease were absent. The diagnosis of incipient locomotor ataxia was made. I had an opportunity to watch this case for about two years, and, as time progressed, nearly all the other symptoms of locomotor ataxia made their appearance. I do not believe that in the literature treating upon this subject sufficient importance has been given to this symptom in the earlier stages of the disease.

The deafness which frequently occurs in those suffering with this disease I believe to be a part of the disease proper, and that it should be considered as forming part of the symptom-complex which characterizes the trouble.

The cause of the deafness is due to a lesion of the auditory nerve in some part of its course, similar to that which affects the roots of the spinal nerves. If these cases in which deafness is present are carefully examined, and the cause of the deafness sought, it will be nearly always found to be of nervous origin; at least, such has been my experience.

An accelerated pulse is another symptom which is usually overlooked. I have reported it present in 25 of the 61 cases here reported. In these 25 cases the pulse was 85 per minute or above. This is probably due to an irritation of the pneumogastric nerve, similar to that which in the spinal nerves produces sensory symptoms.

Another unusual symptom noted in connection with the study of these sixty-one cases was, in eight cases, a dilatation of the pupil, instead of a contraction, as is usually the case. In those cases in which the pupils were dilated, they did not respond to the light, and only very sluggishly to accommodation. In one of the sixty-one cases there was a trace of sugar present in the urine, which has been observed a few times in other cases of this disease.

These cases were seen in all stages of the progress of the disease, some in the first, some in the second, and some in the third stage. As the symptoms change as the disease progresses, the comparison of the symptoms found in any number of cases which may be made by one observer cannot well be compared with an equal number of cases of this disease made by another observer, for the reason that one observer may see more cases in a certain stage of the disease than another; and, secondly, the relative frequency of any symptom or group of symptoms may vary.

The relative importance of the different factors which are supposed to be the cause of the disease is a question with reference to which there is considerable difference of opinion. As nearly as can be determined by clinical observation, the factors which enter into the causation of this disease may be briefly stated as follows: Neuropathic tendencies, syphilis, sexual excesses, exposure to wet and cold, overexertion, mechanical injuries.

Between the opinions of Erb, Möbius, Strümpell, Fournier and others, on the one hand, who regard tabes, in the majority of cases, at least, as purely a post-syphilitic condition, and those of Leyden, Charcot, etc., on the other hand, who disregard syphilis entirely, or give it but a small place in the etiology of tabes, there are many careful observers who hold all grades of opinion between these extremes. Leyden claims that syphilis must be left out of the etiology entirely; while Charcot strongly favored a neurotic heredity, and regarded syphilis of minor etiological importance. The doctrine of the syhilitic origin of tabes is mainly due to Fournier, of Paris (in 1876), and Erb, of Heidelberg (1879). Previous to either of these dates, however, Virchow, Wunderlich and Berger had already expressed themselves as believing that many cases of tabes were of syphilitic origin. In 1883, Erb declared that syphilis was the most important factor in the causation of tabes dorsalis, and that persons not previously infected with syphilis had but the slightest chance of suffering from this disease.

In the third number of his Neurologische Beiträge C. J. Möbius called attention to the changes of opinion of various authors since 1880 in regard to the etiology of tabes. He claims that the number of authors who regard syphilis as an etiological factor is constantly increasing. While Erb, Strümpell and other strong defenders of the syphilitic theory admit that a certain percentage of tabetics can with certainty be excluded, as not having had previous syphilitic infection, Möbius holds the most extreme views, and considers such an infection as the sine qua non for the development of this disease, and believes that Edinger overestimates the importance of excessive function as an etiological factor.

In 1892, Erb found that 89 per cent. of the cases of tabes that he had examined, which included a large number, had previously had syphilis. Fournier states that over 90 per cent. of persons suffering from tabes have had syphilis. Out of 247 cases examined by Hirt, 90 per cent. were syphilitics. Senator gives 70 per cent., Mendel 75 per cent. Gowers, of London, found in his private practice that 58 per cent. of his tabetic patients had previously had syphilis, and he thinks that, could the facts be obtained, two-thirds would be nearer the truth. In Fraenkel's experience, the percentage was 50 7-10; in Gerhardt's, 50; in Dana's, 50. In a minute study of 212 cases, Lagondaky found 42 per cent. syphilitics. Eulenberg gives 36 4-5 per cent. In sixty-eight cases examined by Borgherini, he found that 32 per cent. had previously had syphilis, and 37 per cent. more had suffered from chancroid. A neuropathic taint was found in half his cases. De Werker gives 30 per cent. as syphilitics, while Panas finds that one-third of his cases have previously had syphilis.

Minor shows from his statistics that tabes is much more rare in Russia among the Jews than among other Russians, which is undoubtedly due to the fact that the latter are more or less syphilitic. Magel found 46 per cent. of syphilitics in 1,403 cases. In the experience of Westphal, 14 per cent. of his tabetic patients were syphilitic. In my own experience I have found that 60 per cent. of the cases suffering from tabes dorsalis had previously had syphilis.

Grimm, in the Berlin Medical Gesell., April 11th, 1894, gives the result of his seven years' experience in Japan, syphilis being very prevalent in that country. Of the 13,000 cases which came to his hospital, 1,020 were affected with syphilis. His expectation to find syphilitic tabes was not realized. He observed only five undoubted cases of tabes in Japan, of which only one had a history of syphilis.

Recently, Edinger has advanced a new theory as to the origin of tabes dorsalis, as well as some other diseases of the nervous system. Edinger's theory is based upon the following laws:

1. The healthy normal activity of every organ always causes, or is accompanied by, certain molecular changes which take place in the organ. The organ consequently suffers certain damages, which must be repaired. When the restitution in the organ is equal to the amount of function, the organ is strengthened by functionating; but when not, it undergoes retrogressive changes.

2. A diseased, damaged or weakened tissue soon decays, and gives place to the surrounding healthy tissue, which grows into it and takes its place.

Weigert has proved that so-called hypertrophy, proliferation, interstitial inflammation, etc., are no more than the ingrowth of healthy tissue into the diseased tissue. In the so-called gray atrophies of the central nervous system the nerve tissue is always first diseased, after which it retrogrades, and is finally destroyed by the ingrowth of neuroglia. A degeneration of tissue must follow whenever there is a disproportion between function and restitution to such a degree that the tissue does not return to its former condition. Edinger thinks that, by properly appreciating these facts, many problems may be explained which heretofore have not been fully comprehended. He therefore holds that excessive function is a common cause of tabes, as well as of other diseases of the central nervous system.

Tabes is most frequently observed in persons who overexert their legs, such as military officers, railroad men, etc., while it is seldom found in women of sedentary habits. In these cases the neuromuscular mechanism necessary for the maintenance of the equilibrium of the body and the normal gait is excessively active, and under an uncommonly high pressure. This also explains the rare occurrence of the disease in prostitutes, nothwithstanding they are frequently affected with syphilis, the etiological importance of which Edinger does not seem to underestimate.

The existence of tabetic symptoms in diabetes shows the intimate relation between diseases of the spinal cord and nutrition.

Mechanical injuries are usually considered by most writers on this subject as one of the causes of tabes dorsalis. The cases in which the disease has followed a mechanical injury, or in which there seemed to be a relation between an injury and the development of the disease, are comparatively few, and the evidence that the mechanical injury caused the disease, or was even a factor in the causation, is by no means conclusive.

Hitzig has recently analyzed sixty-six cases in which the cause of the disease was said to be traumatism. In these sixty-six cases, only ten or eleven met the requirements of sound criticism. Hitzig concludes from his study of these cases that the occasional onset of the disease in the injured side, or the preponderance of the symptoms of that side, are not sufficiently constant to warrant a conclusion of the traumatic origin of the disease; but, notwithstanding the great importance of previous venereal infection in most cases of tabes, Hitzig thinks that there are cases which show a distinct relation between traumatism and tabes dorsalis, and which cannot be explained in any other way.

In the February number of the Journal of Nervous and Mental Disease, 1895, Dr. Morton Prince gives a very interesting and valuable article on "Traumatism as a Cause of Locomotor Ataxia." In this article Prince describes quite fully two illustrative cases which show the liability to error in arriving at a conclusion in this matter, when the statements of the patient are taken as the basis for an opinion. Dr. Prince thinks—and the point is well illustrated in the two cases he reports—that a person may have locomotor ataxia for some months, or even several years, without knowing it, or knowing that anything is specially wrong with him. Especially is this apt to be the case when the sensory symptoms are in abeyance. In view of this fact, Prince insists that the following rules must be rigidly adhered to in examining the evidence afforded by alleged individual cases:

"1. The subject must have been proved free from tabes, either immediately before or immediately after an accident.

"2. The subject must be shown not to have been exposed to other known causes, as syphilis, for example.

"3. The traumatism must have been of a nature to produce a physical or psychical impression of an appreciable degree, and not such a one as people are frequently exposed to without suffering afterward from tabes, e. g., the extraction of a tooth or a mild bruise.

"4. The symptoms must have made their appearance within a reasonable time after the accident—at least within a year.

"5. The diagnosis must have been established beyond a reasonable doubt."

In this article Prince analyzes critically the evidence which is given in cases thus far reported, in which traumatism was the supposed cause. He divides these cases into three classes.

The first class, consisting of twenty-two cases, he considers inadmissible on account of the triviality of the injury, the pre-existence of syphilis, long interval between injury and onset of symptoms, doubtful diagnoses, etc.

The second class of cases, consisting of six, are those which cannot be excluded, but which, from various circumstances, are questionable in evidence.

The third class are those in which the disease was apparently caused by traumatism. Of these, there are twelve cases. From those studied, Prince gives the following as the result of his investigation of this subject:

"Taking all the facts above collated into consideration, it would seem that the current view that locomotor ataxia may be caused by traumatism, per se, irrespective of direct lesion of the cord, is not sustained by the evidence thus far adduced. If such a relation exists, further evidence is required before it can be accepted. It would seem to be more probable, aside from mere coincidence, that when a sclerosis of the posterior column develops after a traumatism, the subject was already doomed to this condition, the process having already begun, and that the traumatism, at most, but accelerated the symptoms, and, possibly, the anatomical process."

In the writer's opinion, there are two things, or conditions, necessary to the development of this disease in any individual. One of these is an organic predisposition to the disease; that is, a neuropathic condition—a low resistance in the nerve elements. The other essential condition is the presence in the blood and tissues of a toxin, which, in a very large proportion of cases, is of syphilitic origin. This toxin, in the case of syphilis, is constantly formed in the body after it once becomes infected, and keeps up a continuous chronic intoxication for a number of years, until finally the vitality of the tissues is overcome, and the symptoms of tabes begin to appear.

Other infections, both acute and chronic, have been known to cause this disease. Cases are on record in which tabes developed after infection from tubercular disease and also from leprosy. These infections, like that of syphilis, are chronic; that is, the germs of the disease remain in the system for a long period of time, and, as the result of the life and activity of these germs, ptomaines are constantly formed, and consequently the system is kept in a state of intoxication for a period of months, or even years. The disease may also follow or accompany infections of acute diseases, such as diphtheria, typhus and typhoid fever. It is a well known fact that in many cases of diphtheria there is a post-diphtheritic paralysis. This is quite frequently supposed to be due to a neuritis which is the result of the action of diphtheria toxins upon the nerve fibres. This same poison may, and does, in some cases at least, produce tabes. In my own experience I have seen one well developed case of locomotor ataxia (tabes dorsalis) follow an attack of typhoid fever.

There is, however, an essential difference between cases of tabes resulting from chronic infection like syphilis or tuberculosis, and those of an acute infection like diphtheria, typhoid fever, etc. In the first instance, the disease, in the large number of cases at least, is progressive, which is a natural result if the body is kept constantly intoxicated by poisons that are formed by the infection of syphilis or tuberculosis. In the second instance, that is, where the disease develops from an acute infection, it becomes, after a period at least, regressive; it does not continue to progress as in the first instance. The best reason I can give for this difference is that in these cases poisons are formed in the body only for a limited period of time. The germs of diphtheria and typhoid fever remain in the system but a few weeks, and consequently the poisoning continues only for a limited space of time.

The question will naturally arise in the minds of some, whether these cases of so-called tabes dorsalis following acute infections, such as typhoid fever and diphtheria, are really true cases of tabes dorsalis, or whether the ataxic symptoms are due to a multiple neuritis, which frequently follows these infectious diseases. To this point I have but to answer that cases of tabes dorsalis have been reported by those of experience and careful observation. In my own personal experience I have seen only two cases of this kind, which appeared to me to be cases of tabes dorsalis, and not multiple neuritis.

It is as unreasonable as it is unscientific, in the light of modern pathology, to suppose that a man in the prime of life, thirty years of age, for instance, should be attacked with a disease like locomotor ataxia, which constantly progresses and becomes more severe in character and wider in extent, causing the individual to stagger through life, and finally totter into the grave, except there be in the body a cause that is constantly present and active. Certainly no good reason can be given why in a man in apparently good health there should begin to be formed on the posterior columns of the spinal cord a strip of sclerosed tissue, which constantly extends its borders, unless, as said before, there exists in the body a cause that is constantly acting. To say that this might be caused by perverted action on the part of the tissues—a sort of habit, as it were—is a very poor and unsatisfactory explanation. It does not give any reason why the disease should constantly progress as it does.

Besides the infections above referred to, chemical poisons may cause this disease. It is well known that poisoning from ergot, also from lead and mercury, is frequently followed by symptoms of tabes dorsalis. Besides these, poisoning from substances formed within the body in gouty, rheumatic and diabetic diatheses is sometimes followed by symptoms of locomotor ataxia. Locomotor ataxia may also follow pernicious anæmia.

I do not believe that mechanical injury, exposure to wet or cold, overwork, or sexual excesses without infection can be regarded as an exciting cause of tabes dorsalis, per se. None of these influences, acting alone or in conjunction, could cause a bundle of nerve fibres to degenerate, and the adjacent connective tissue to increase until a well defined, triangular band of sclerosed tissue is formed for a greater or less distance along the posterior part of the spinal cord. That these agents have some influence in exciting the disease in many cases is granted, but their action is in lessening the vitality of the nerve elements, and preparing them for another more active agent, which I believe is always a poison from some of the sources previously mentioned. The essential thing is a toxin in the blood, which may be a ptomaine, a leucomaine, or a chemical poison, either organic or inorganic.

Much valuable work has been done recently with reference to the pathogenesis of tabes. The old theory, which considered the primary lesion in tabes as a degeneration of the posterior columns of the spinal cord, will, in the light of recent research, hardly hold. That there is a degeneration of the nerve fibres of the posterior columns of the cord is a well established fact; but is this primary? Or is it the result of another lesion, which, at least in a casual relation, antedates it?

Rindfleisch ascribes the origin of tabes to a slightly progressive inflammation of the pia mater, which by reason of tissue continuity gradually extends to the connective tissue of the cord, and there forms an interstitial myelitis, which results in a secondary degeneration of the nerve fibre of the cord. Obersteiner and Redlich have recently advocated a modification of this idea. They have attempted to determine the exact starting point of the degeneration. By making oblique sections of the spinal roots in the direction in which they enter the cord, they have found that, normally, the roots are constricted by a circular band of connective tissue of the pia mater.

In the early stages of tabes, the roots of the spinal side of the restriction are found to be degenerated; the cause of this degeneration is a hyperplasia of the connective tissue, which forms the constriction. This presses on the nerve fibre, and leads to its degeneration. The reason that the nerve fibres in Lissauer's boundary zone of the spinal cord differ so in the disease is that in the spinal roots these fibres are located in the periphery, and consequently suffer most from the constriction. Nageotte, in the Société de Biologie, November 10th, 1894, gives his views of the primitive lesion of tabes. He believes that the initial lesion of tabes is a perineuritis, which affects the posterior spinal roots between the spinal ganglia and the point where the roots enter the subarachnoid space. The perineuritis at first partakes of the nature of an embryonic process, but later is fibrous in character. Following the inflammatory process is a degeneration of the root fibres, which extends into the posterior columns of the cord. The anterior as well as the posterior spinal nerve roots are affected by the inflammation; but, as they seem to have greater resistance, the motor nerve fibres are less liable to degenerate.

Prof. Leyden, as early as 1863, expressed the opinion that the lesion in tabes begins in the posterior roots, and that the degeneration in the posterior columns of the cord is secondary. The researches of Obersteiner, Redlich and Nageotte, previously referred to, as well as those of the French neurologists, Marie and Dejerine, concur in the opinion expressed so long ago by Leyden. As to the primary lesion in tabes, Marie, however, holds that it is in the ganglia of the roots, and not in the root fibres.

It seems quite well established, therefore, that the disease begins in the posterior spinal roots, either in the nerve fibre or the ganglia, the nature of the lesion being at first inflammatory, and affecting the connective tissue, and, later, degenerative, affecting the nerve fibres.

The nerve fibres which form the posterior column of the spinal cord are but the extension of those which make up the posterior spinal roots.

A lesion affecting the fibres of the posterior spinal roots would likewise affect those of the posterior columns of the cord. A lesion of the posterior roots would interfere with the nutrition of the fibres of the posterior columns of the cord by cutting them off from their centre of nutrition, which is the spinal ganglia of the posterior roots.

With reference to the treatment of this disease, space will not allow a minute description. I can make only a few general suggestions. My own practice for some time past has been to direct the treatment along two lines, viz., (1) to eliminate poisons that are in all probability being constantly formed; (2) to improve the general health and nutrition of the patient, special treatment being directed to the seat of the lesion.

The first of these lines of treatment, that is, the elimination of the poisons from the body, I believe can be best accomplished by the free use of water internally. I usually instruct my patients to drink from five to seven pints of water daily, and to take it in small quantities and at short intervals between meals. This flushes the tissues, and keeps the poisons that may be in the body in solution, thus favoring their elimination. It also increases the activity of the eliminative organs. In many cases it is best to have the patient drink the water hot. Besides this, the use of warm baths, particularly the electric light bath, which is usually very agreeable to these patients, is to be highly recommended. This does good, not only by the elimination of poisons, but also by relieving the distressing pains, which are so troublesome in this disease. By correcting all bad habits, prohibiting the use of tobacco and alcohol, and placing the patient on an aseptic diet, the further intoxication of the body from without becomes almost impossible.

The second indications for treatment are best met by the proper use of hydrotherapy, electricity in its various forms, massage and other manual and mechanical movements, including suspension treatment. I am satisfied from the results of the treatment in a large number of cases of this kind that a great deal more can be done for patients suffering with this disease than is usually supposed. I do not myself lay claim to any superior wisdom or extraordinary skill in managing these cases; but I believe from my own experience that when they can be put under proper conditions—the daily life of the patient controlled, and the treatment above indicated intelligently applied—as much can be accomplished in this malady as in many other diseases affecting other parts, which are generally considered less intractable.

It is certainly unjust to tell a man suffering from locomotor ataxia, in the first or second stage of the disease, that nothing can be done for him; I have had many cases concerning which such statements had been made by physicians, and yet, after taking the course of treatment as indicated, they have materially improved, the disease being checked in its onward progress, and the patient put in a position where he could be of practical service to himself and to his friends. And this is as much as, or more than, is done in the treatment of numerous other diseases which are not supposed to be as formidable as tabes dorsalis.

221. EIN BEITRAG ZUR PATHOLOGIE UND PATHOLOGISCHEN ANATOMIE DER TRAUMATISCHEN RÜCKENMARKSERKRANKUNGEN (SOGENANNTE HÄMATOMYELIE, SECUNDÄRE HÖHLENBILDUNG. (A Contribution to the Pathology and Pathological Anatomy of the Traumatic Diseases of the Spinal Cord, etc.) Lax and Müller (Deutsche zeitschrift für Nervenheilkunde, 12, 3 and 4).

A man became paralyzed in all his limbs immediately after a fall. Complete anæsthesia reached as high as the axillæ. The movements of the head and neck were not interfered with. Cerebral or bulbar symptoms were not noted, and consciousness was preserved even immediately after the accident. Motion in the shoulders and elbows gradually became possible, but the hands remained much paralyzed. The complete anæsthesia gradually changed to analgesia and thermoanæsthesia. Tactile sensation was regained in the entire body. A gradually developing atrophy of the interosseous muscles of the hands and of the extensors of the forearms was noted. The muscles of the lower extremities became rigid, and all the tendon reflexes were exaggerated. Death occurred three years after the fall.

The vertebral column was intact. A cavity was found in the posterior part of the cord, and was limited to the fifth cervical segment. The lateral columns were sclerosed. Bands of tissue, consisting of glia fibres, vessels and medullated nerve fibres, were found within the cavity, and bore much resemblance to peripheral nerves. Secondary ascending and descending degeneration was noted. No remains of a former hemorrhage could be observed, but the cavity was believed to be due to the absorption of a hemorrhage and disorganized nerve tissue. A recent case of hæmatomyelia resulting from fracture of the vertebræ is also noted. The writers believe that the lower cervical region is a frequent seat of spinal hemorrhage. They think that a force which is sufficient to cause hemorrhage causes injury also of the nervous tissue, not depending directly on the hemorrhage. The case is important, as showing the development of syringomyelia after trauma. SPILLER.